

Conclusions. The isolated cerebroside was not the customary kerasin; it contained *d*-glucose in place of the usual *d*-galactose component, as proven by fermentation, optical rotation and reducing equivalents. Halliday, *et al.*, suggested that the cerebroside isolated by them might represent an anomaly of carbohydrate metabolism. Our results, and those recently reported by Klenk and Schumann,⁹ indicate that synthesis of a glucoside type of splenic kerasin is a frequent occurrence in Gaucher's disease.

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Electrical Properties of Tissues in Shock Therapy.

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A lack of knowledge of the exact treatment current has hindered the standardization of electrical shock therapy. This has been especially true because the treatment current does not appear to follow "Ohm's law"—that is, the current is not equal to the treatment voltage divided by the measured (d-c) resistance between the electrodes, but usually is considerably higher. Also, the resistance varies in what has appeared to be an "unpredictable" manner. For these reasons, an *a priori* selection of the treatment current has appeared to be impossible.

It is, of course, the passage of the electric current which is responsible for the convulsive shocks, rather than the applied voltage of itself, so that dosage standardization must be on the basis of the former. The voltage required to obtain a given current will depend upon many factors, such as the thickness of the skull, area of electrodes, condition of the skin, etc. Obviously a rational prescription of dosage requires some method of taking these factors into account. This was recognized even in the original work of Cerletti and Bini (1938), who employed a d-c resistance measurement as mentioned above. However, this bears no relationship to the effective resistance during treatment.

A study of the electrical properties of the tissues involved offers an explanation of the apparently anomalous behavior of the resistance. This has made possible the accurate preselection of the treatment current used in shock therapy.

⁹ Klenk, E., and Schumann, E., *Z. physiol. Chem.*, 1940, **267**, 128.

Method. Two electrodes were placed on the subject's head, using electrode jelly, as for shock treatment. The alternating current impedance between these was measured at frequencies of from 50 to 15,000 c.p.s. by means of an impedance bridge.* The very low current employed produced no stimulation. The alternating current impedance of tissue may be separated into two components: the resistance or energy dissipating portion; and the reactive, or energy storing portion. The latter results from the polarizability of living tissue.

Considerable information concerning the electrical behavior—and also at least to some extent the structure—of tissue may be obtained by plotting the "impedance locus" (Cole and Curtis¹). In this, the reactance is plotted against the resistance for each frequency at which the impedance is measured. Proper analysis will then frequently allow the derivation of an "equivalent electrical network," in which the electrical properties of the tissues are represented by a combination of ordinary electrical components, such as resistors and condensers.

Electrical Results. A typical impedance locus as obtained in the above measurements is illustrated in Fig. 1. An analysis of the figure shows that these properties may be approximated by the network of Fig. 2, with R_1 100 ohms, and R_2 500 ohms. C is the element of the network introducing the reactance, and represents the membrane polarizability. It is a condenser of about $0.4 \mu\text{fd}$ capacity and 62° phase angle.

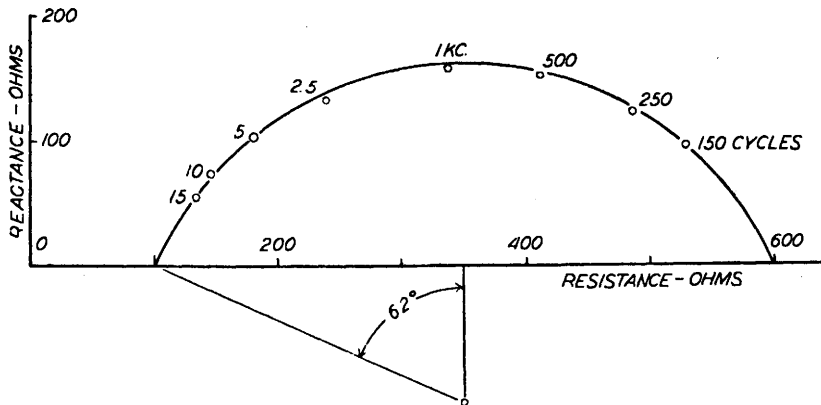


FIG. 1.
Impedance locus of electrodes.

* Offner Electronics, Type 800.

¹ Cole, Kenneth, and Curtis, H. J., *Cold Spring Harbor Symposium*, 1936, 4, 73.

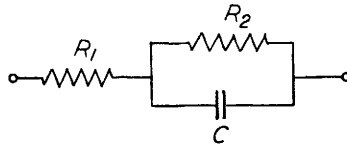


FIG. 2.
The equivalent circuit.

As direct currents cannot pass through a condenser, it is apparent that the d-c resistance of the network will be $R_1 + R_2$, or 600 ohms. This is the high resistance intercept of the impedance locus of Fig. 1—the “zero frequency extrapolation.” Very high frequencies will, on the other hand, pass through C almost unimpeded, so that no current will flow through R_2 . The effective network resistance will then be R_1 , which is the low resistance intercept of the locus—the “infinite frequency extrapolation.”

It has been shown (Cole and Curtis²) that electrically stimulated tissues lose almost completely their polarizability; that is, they become almost completely permeable. In the above network, this may be represented by R_2 falling to such a low value that it effectively short-circuits the condenser C . The measured resistance of the network at all frequencies will then be only slightly greater than R_1 . Now the current used in shock therapy is of quite great strength, and it must stimulate the tissues carrying the bulk of the current. Thus during the passage of the treatment current, the resistance of R_2 will fall to a low value, and the effective resistance to the passage of the treatment current will be only slightly greater than R_1 . It is apparent, however, that the *effective* resistance of the patient can be determined beforehand by use of a high frequency test current, rather than the direct current originally used. The following relationship may be stated: The measured electrode resistance before treatment, using a low intensity, high frequency testing current, should equal the resistance to the passage of the high intensity, low (60 c.p.s.) frequency treatment current.

The above relationship was used in setting the dosage in shock therapy. A 7000 cycle testing current is first passed through the electrodes on the patient. The voltage is adjusted until this testing current is a fixed fraction (about one ten-thousandth) of the desired treatment current; the treatment voltage to be given is simultaneously raised proportionately. When the shock is given, the treatment current should then bear this same relationship to the test current, and the

² Cole, Kenneth, and Curtis, H. J., *J. Gen. Physiol.*, 1939, **22**, 37, 649.

test current meter may thus be calibrated directly in terms of the treatment current.

Both because of its low intensity and because its frequency is remote from the pararesonance frequency of nerve cells (Monnier³), the test current cannot be felt by the patient.

The fact that the d-c resistance between the electrodes is almost meaningless became obvious during the impedance measurements; it was found that it could be changed at will by varying the technic used in electrode application. Such variations did not, however, appreciably affect the high frequency resistance. Nor has experience in administration of shocks indicated that they affect the value of the treatment current.

Convulsive shocks were administered to a number of patients with the instrument, as listed in Table I. In every instance the treatment current was very nearly equal to the value predicted by the test meter—always within 5%. The rather large internal stabilizing resistance of the instrument tends to obscure small differences between the patient's resistance to the testing and treatment currents. The results, however, confirm the conclusion that the effect of the tissue polarizability is largely eliminated by the flow of the treatment current.

The advantages of the use of this technic in the administration of electric convulsive shocks appears to be two-fold: it allows the value of the treatment current to be set at the value desired for the treatment; and the current values used by different workers can be compared. The voltage required to obtain a given current will vary with the exact form of electrode employed, as well as anatomical differences between patients. Thus it would be expected that considerable variation in voltage values would occur from subject to subject, as well as between different workers for the same subject. On the other hand, current values should be more nearly constant. It has, in fact,

TABLE I.

Patient No.	Test current, ma.	Treat. current, ma.
1	380	380
2	380	380
3	330	320
4	400	400
5	450	450
6	190	180
7	450	455
8	450	450
9	330	330

³ Monnier, A. M., *L'Excitation Electrique des Tissus*, Hermann et Cie, Paris, 1934.

been found that the large percentage of subjects will receive convulsions with currents of from 400 to 450 ma, applied for 0.2 or 0.3 seconds.

The writer wishes to thank Drs. E. Dombrowski and J. V. Edlin of the Chicago State Hospital for their coöperation in the test of this instrument, and Dr. R. W. Gerard for assistance in preparation of the manuscript.

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Influence of Hyperpyrexia on Ascorbic Acid Concentration in the Blood.

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It has been established^{1, 2, 3} that the plasma ascorbic acid concentration during the course of an infectious fever is lowered, as a consequence, the therapeutic procedure of supplementing the diet of such patients with ascorbic acid is receiving increased attention.

This investigation was undertaken to ascertain whether an artificial fever produced by physical means also produced a lowering of the plasma ascorbic acid. When this work was initiated no such study had been reported; but in the meantime two somewhat similar investigations have been reported.^{4, 5} In addition, Rhinehart's^{6, 7} association of low plasma ascorbic acid levels with the etiology of rheumatic disease further augmented our interest, inasmuch as our patients had been afflicted with this disease.

Method. Seventeen patients suffering from chronic arthritis were subjected to hyperpyrexia. The body temperature was elevated and maintained at 104° to 104.6° F (rectal) for a period of 4 hours, following which it was permitted to return to normal. A combination of fever cabinet and inductotherm was used to elevate the patient's temperature. Treatments were given once weekly.

1 Farmer, C. J., *Quart. Bull. Northwestern Univ. Med. Sch.*, 1940, **14**, 220.

2 Wolfer, J. A., *Surg. Clin. N. A.*, Chicago Number, Feb., 1940.

3 Wolfer, J. A., *Surg. Gynec. Obst.*, 1939, **69**, 745.

4 Daum, K., Boyd, K., and Paul, W. D., *Proc. Soc. Exp. Biol. and Med.*, 1939, **40**, 129.

5 Zoak, J., and Sharpless, G. R., *Proc. Soc. Exp. Biol. and Med.*, 1938, **39**, 233.

6 Rhinehart, J. F., *Ann. Int. Med.*, 1935, **9**, 586.

7 Rhinehart, J. F., *Ann. Int. Med.*, 1935, **9**, 671.